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The Clinical Course of Multifocal Atrial Tachycardia in Infants and Children

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OBJECTIVES This study outlines the clinical course, treatment and the late outcome of infants and children with multifocal atrial tachycardia (MAT).

BACKGROUND Multifocal atrial tachycardia is defined by three distinct P-waveforms, irregular P-P intervals, isoelectric baseline between P-waves and rapid rate on an electrocardiogram. Several smaller prior reports have described pediatric patients with MAT, but their long-term outcome has not been fully assessed.

METHODS The clinical records, echocardiograms and long-term follow-up of patients with MAT were reviewed and compared to previous reports of MAT.

RESULTS Fourteen boys and seven girls (median age 1.8 months) presented with MAT. At diagnosis, six patients had respiratory illness, of whom two were critical. Ten were asymptomatic. Seven patients had structural heart disease (SHD), one of whom died. Four of 15 patients (27%) with echocardiograms had diminished ventricular function. Ventricular rates were 111 to 253 beats/min (mean 181 beats/min). Median duration of the arrhythmia was 4.9 months (mean 6.7 months). Electrical cardioversion was attempted in 4 patients without success and 15 patients received antiarrhythmic medication. Seventeen patients were followed for a mean of 60 months. Four patients were lost to follow-up. There were no late arrhythmias.

CONCLUSIONS The majority of children with MAT are healthy infants under one year of age; a few may exhibit mild to life-threatening cardiorespiratory disease. Less often, MAT accompanies SHD. Mild ventricular dysfunction may be observed in the presence of MAT, but symptoms are few and resolution is generally complete. Response to antiarrhythmic agents is mixed, and cardioversion is of no avail. Finally, long-term cardiovascular and developmental outcome depends principally on underlying condition; for otherwise healthy children, it is excellent.
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Multifocal atrial tachycardia (MAT), also known as chaotic atrial rhythm (1), is a rare tachyarrhythmia in infants and children, accounting for <1% of supraventricular tachycardia (SVT) seen by the Pediatric Electrophysiology Service of the University of Michigan Health Systems over the past 20 years. The condition is characterized by the electrocardiographic (ECG) finding of: 1) multiple (at least three) distinct P-wave morphologies; 2) irregular P-P intervals; 3) isoelectric baseline between P-waves; and 4) ventricular rate >100 beats/min (2) (Fig. 1). Similar to the more common forms of SVT seen in childhood, MAT is very rapid—atrial rates up to 400 beats/min and ventricular rates at 150 to 250 beats/min—but unlike most SVT, it is markedly irregular. Pauses may be observed following blocked premature atrial impulses. At high atrial rates, aberrant ventricular conduction might resemble nonsustained ventricular tachycardia. Although MAT might be the predominant rhythm for weeks or months, in most children the irregular, rapid rate alternates with periods of normal sinus rhythm.

Often, MAT is diagnosed in elderly adults, particularly in those with chronic obstructive pulmonary disease (3,4). Although one report associated MAT in children with hemodynamic collapse, most investigators have described it as usually causing mild symptoms, and having few sequelae (1,5–11).

The purpose of this report was to outline the presentation, course, treatment and late clinical outcome in 21 infants and children with MAT seen at our institution, along with a summary of similar patients reported elsewhere.

METHODS

Using departmental patient databases, 21 patients meeting the ECG criteria (2) for MAT presenting during the past two decades were identified. Clinical history, physical examination and other clinical data were reviewed. Diminished left ventricular function was defined as a shortening fraction below 0.30 or a two-dimensional ejection fraction <0.50 measured by echocardiogram. When those measurements were not obtained, the reported subjective description of diminished function was used. In 19 patients, follow-up Holter monitor tracings or electrocardiograms (ECGs) provided confirmation of complete resolution of MAT. Particular attention was paid to the follow-up cardiac status, recurrence of MAT or other arrhythmias, evidence of cardiac dysfunction or other significant health problems.

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Abbreviations and Acronyms

ECG = electrocardiogram, electrocardiographic
HOCM = hypertrophic obstructive cardiomyopathy
MAT = multifocal atrial tachycardia
RSV = respiratory syncytial virus
SHD = structural heart disease
SVT = supraventricular tachycardia

Chi-square analysis was used to evaluate differences between patient groups. A p value of ≤0.05 was taken to denote statistical significance. The Institutional Review Board for Investigation Using Human Subjects, the University of Michigan Health Systems, approved this study.

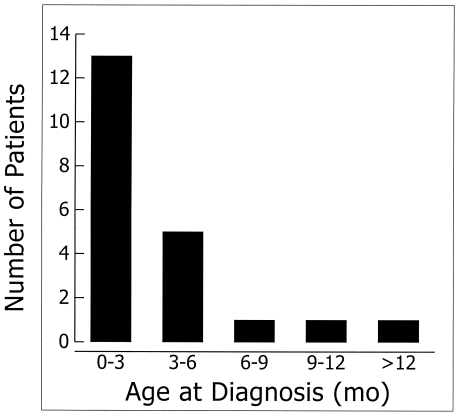

Figure 2. Histogram demonstrating distribution of patients by age at diagnosis of multifocal atrial tachycardia. Most children diagnosed with multifocal atrial tachycardia are infants.

RESULTS

Twenty-one children with MAT were identified: 14 boys and 7 girls. The median age at diagnosis was 1.8 months (Fig. 2) (mean 6.5 months; range 0 days to 7.3 years). Six patients were diagnosed at birth (in 3 patients, irregular rhythms were observed on fetal monitors), and 20 (95%) were diagnosed before one year of age. Six patients had symptoms of intercurrent respiratory illness at diagnosis: two with nonspecific upper respiratory infections; one with croup; one with bronchomalacia exacerbated by postoperative atelectasis; one with antigen test-positive respiratory syncytial virus (RSV) pneumonia, and one respiratory viral syndrome with myocarditis. These last two were critically ill. Five other patients had symptoms or signs (e.g., irritability, tachypnea) possibly due to cardiovascular compromise. Ten patients (48%) were asymptomatic; the arrhythmia was identified in three patients during routine examination, one by a home monitor, three on routine monitoring during medical procedures, and three on fetal monitors; in one patient the method of detection is unknown. Among the 15 patients evaluated by echocardiogram, 4 had quantitative (n = 3) or subjective (n = 1) evidence of diminished ventricular function (Table 1), one of whom had structural heart disease (SHD). Duration of the arrhythmia, documented in 19 patients, was a median of 4.9 months (mean 6.7 months; range 8 days to 18.5 months) (Fig. 3). For the 14 patients in whom it could be accurately calculated, the median interval from detection of an irregular cardiac rhythm to precise diagnosis was four days (mean 30 days; range 0 to 238 days).

Although detection of the arrhythmia did not lead to the diagnosis of SHD in any patient, seven (50%) had anatomic abnormalities. One had Klippel-Feil syndrome and repaired tetralogy of Fallot; one had Noonan syndrome and recent repair of pulmonic stenosis and atrial septal defect; one had myocarditis; one suffered familial hypertrophic obstructive cardiomyopathy (HOCM), and one had HOCM, double-outlet right ventricle and single coronary artery. The patient who died had heterotaxy syndrome with asplenia, atrioventricular septal defect, pulmonary atresia and growth failure. He had undergone aortopulmonary shunt placement. The seventh patient with heart disease was diagnosed with aortic coarctation 18 months after resolution of MAT. No echocardiogram had been performed, nor had a murmur, differential pulses or blood pressure gradients been observed at his initial presentation.

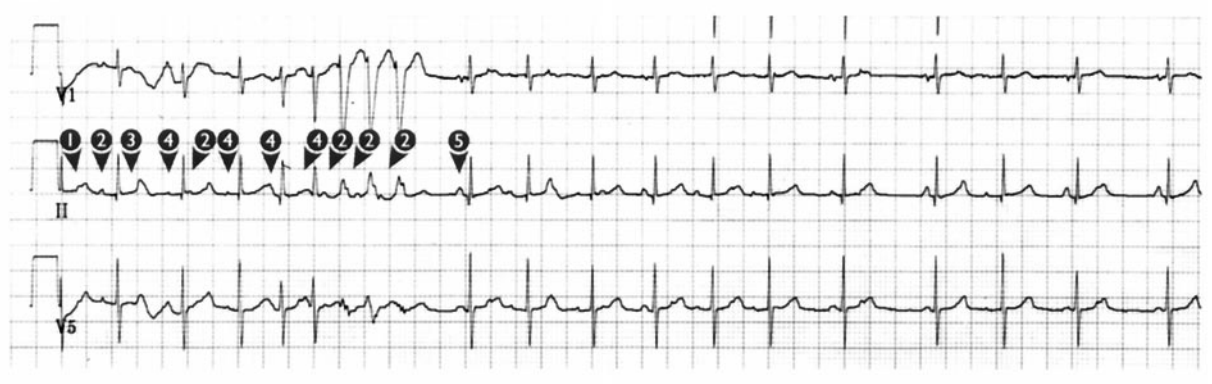

Figure 1. Electrocardiogram (from patient 6) demonstrating typical characteristics of multifocal atrial tachycardia, with five different P-wave morphologies **(arrows)**, irregular, rapid atrial rhythm and variable ventricular conduction.

Fifteen patients had echocardiograms as part of their evaluation; 11 demonstrated normal cardiac function and chamber dimensions. Of these 11, 3 had normal ventricular function and cardiopulmonary symptoms, 4 patients had abnormal function but no symptoms, and 4 had normal function and no symptoms. Of the five patients who had both SHD and an echocardiogram while in MAT, one demonstrated diminished function. No relationship was seen between diminished left ventricular function and cardiorespiratory symptoms ($\chi^2 = 1.0$; $p = 0.31$). The mean ventricular rate at diagnosis was 181 beats/min (range 111–253 beats/min). There was no significant difference in the mean ventricular rate between patients with and without diminished left ventricular function (176 vs. 196 beats/min; $p = 0.45$).

Treatment, initiated either at the referring institution or at our own institution, is summarized in Table 2. Fifteen patients (71%) received at least one antiarrhythmic agent. Four patients underwent attempts at direct current or overdrive pacing cardioversion, but without success. Ventricular rate-slowing effects of agents such as digoxin were noted but could not be verified.

Follow-up data were available for 17 patients; mean follow-up interval was 60 months (range 8 to 216 months). Four patients were lost to follow-up. Three of the four children (except for Patient 16, with myocarditis) with abnormal ventricular function in the presence of MAT had normal function upon repeated imaging after cessation of MAT. In the follow-up group ($n = 17$), three patients are taking medication; two patients with HOCM are taking verapamil (Patients 12, 17) and one (Patient 10) is taking digoxin for diminished right ventricular function after repair of tetralogy of Fallot. The one death occurred in the patient with heterotaxy syndrome (Patient 21) while hospitalized for respiratory distress. Receiving digoxin and amiodarone and nearing discharge, he developed progressive, severe bradycardia during feeding and could not be resuscitated. Monitor tracings demonstrated sinus rhythm for six days prior to his death. In the follow-up group, three patients (Patients 5, 10, 20; 14%) have delayed psychomotor development; of these, one (Patient 20), has Noonan syndrome and two (Patients 5, 10) are hearing-impaired. All other patients are alive and well, without any cardiovascular symptoms, and at an age-appropriate developmental level at last follow-up. The 16 living patients in the follow-up group are free of tachyarrhythmia.

When the published reports of MAT in children (Table 3) are considered together with those in this report ($n = 105$), mortality was 11% during the period of follow-up. In the 33 (31%) with heart disease, mortality was 24% (8 of 33), and in those without heart disease mortality was 4% (3 of 72; $\chi^2 = 9.7$; $p = 0.001$). Male patients ($n = 68$), who comprised 65% of the total population, had a mortality rate similar to that of female patients ($\chi^2 < 0.01$; $p = 0.99$).

DISCUSSION

This report of 21 children with MAT (also known as chaotic atrial rhythm or chaotic atrial tachycardia) confirms previous studies with regard to their age range, clinical context and outcome and extends the duration of follow-up (1,5–9,12). Seen in a range of clinical settings in our study, MAT was self-limited and nonrecurring. Of the 19 children in whom arrhythmia duration was known, 10 reverted permanently to sinus rhythm within five months (Fig. 3). Sixteen of 17 patients demonstrated no cardiac symptoms or arrhythmia in long-term follow-up (mean 60 months). The one exception is Patient 10, who has not had a tachyarrhythmia but has required a pacemaker for transient complete heart block observed during anesthesia induction.

Mortality. Yeager et al. (11) reported on four infants presenting with MAT, three of whom died two to five months after diagnosis. One infant had poorly controlled ectopic atrial tachycardia prior to the diagnosis of MAT, marked mitral valve abnormality and pathologic findings of myocarditis at autopsy. One was diagnosed with MAT at six weeks of age, but had normal ECGs two weeks later. He died in his crib at seven months. The third patient who died had multiple craniofacial anomalies and tetralogy of Fallot. At three months of age he developed respiratory distress following a feeding, and died before an ECG could be obtained. Chest radiograph demonstrated bilateral lung infiltrates. Although the presence of MAT could not be confirmed at the time of death in two of these three patients, the investigators postulated that the arrhythmia might have been related to these deaths. One of our patients (Patient 6), who had severe respiratory insufficiency but normal cardiac function by echocardiogram, required cardiopulmonary resuscitation during his illness but survived. The three-month-old boy in our series with complex heterotaxy syndrome (Patient 21) represents the sole death in our series. The severity of this patient's anatomic defect, the absence of arrhythmia at the time of his death and the infrequency of death in reports of MAT especially in the absence of SHD suggest that MAT per se is not strongly linked to patient mortality. Of the 105 reported patients, the significantly higher mortality in those with SHD than in those without (24% vs. 4%) underscores the important influence of the underlying heart disease. Similarly, 14 of our 21 patients had anatomically normal hearts (i.e., neither acquired nor congenital heart disease), suggesting that MAT does not require an abnormal heart for its initiation or persistence.

Electrophysiologic mechanism. The electrophysiologic mechanism or mechanisms underlying MAT remain unknown. Most investigators have postulated that the atrium is activated at a rapid rate by impulses from multiple automatic foci. This accounts for the many P-wave morphologies, their irregularity and their frequency (Figs. 1 and 4A,B). Additionally, as with ectopic atrial tachycardia, which arises from a single automatic focus, it would explain MAT's poor response to cardioversion. In a dog model, rapid stimulation of the right vagal nerve produced multi-

Table 1. Patient Characteristics

Patient No.	Age	Gender	Symptoms While in Tachycardia	Medical History, Intercurrent Illnesses, Chronic Pathology
1	3 mo	F	Rhinorrhea	Ex-23 wk triplet, mild URI
2	1 mo	M	None	None
3	1 mo	F	None	None
4	2 mo	M	Increased fussiness	Diarrhea × 2 days, afebrile, mild URI
5	Newborn	M	None	Prematurity, dysmorphic, deaf, growth and developmental delay
6	2 mo	M	Respiratory distress, thick rhinorrhea	Saggital sinus thrombosis at birth, RSV pneumonia, respiratory arrest
7	Newborn	F	None	None
8	3 mo	M	Mild irritability and conqestion	Mild URI, normal development
9	Newborn	M	Tachypnea	Coarctation, verrucous nevus, small CCAM
10	11 mo	M	Stridor with mild-moderate respiratory distress	Klippel-Feil syndrome, repaired TOF, Sz, developmental delay, croup
11	2 mo	M	None	History of urgent cesarian section for arrhythmia
12	Newborn	F	Hypotonia, tachypnea	Birth asphyxia due to shoulder dystocia, HOCM, DCRV, ASD, single RCA, Noonan syndrome
13	1 mo	F	None	None
14	Newborn	F	None	None
15	4 mo	M	None	None
16	3 mo	M	Marked respiratory distress, rhinorrhea, fever	Myocarditis, URI progressing 2 weeks before presentation
17	7 yr	F	None	Familial HOCM
18	1 mo	M	None	None
19	Newborn	M	Fussiness	None
20	8 mo	M	Tachypnea	Noonan syndrome, ASD, PS, s/p valvulotomy, bronchomalacia, pleural effusion, LUL atelectasis
21	9 days	M	Irritability, variable perfusion, mild hypotension	Heterotaxy, AVSD, pulmonary atresia, TAPVR, single ventricle, s/p central shunt, PA augmentation

ASD = atrial septal defect; AVSD = atrioventricular septal defect; CoA = coarctation; CCAM = congenital cystic adenomatoid malformation; DCRV = double-chamber right ventricle; EF = ejection fraction; fx = function; HOCM = hypertrophic obstructive cardiomyopathy; LUL = left upper lobe; LV = left ventricle; LVEDD = LV end diastolic dimension; LVSD = LV systolic dimension; MR = mitral regurgitation; n/a = information not available; PA = pulmonary artery; PI = pulmonary insufficiency; PS = pulmonary stenosis; RCA = right coronary artery; RSV = respiratory syncytial virus; RV = right ventricle; SF = shortening fraction; s/p = status post; Sz = seizure disorder; TAPVR = total anomalous pulmonary venous return; TOF = tetralogy of Fallot; TR = tricuspid regurgitation; URI = upper respiratory infection.

focal atrial depolarizations and sinus pauses, raising the possibility that autonomic influences might be responsible for MAT (13). Another possible mechanism is that the varied P-wave morphologies seen in MAT may not represent impulses of multifocal origins, but rather varied propagation of impulses originating at a single focus. Bevilacqua et al. (14) recently described a four-month-old with MAT (the investigators' term was "chaotic atrial rhythm") and severely depressed ventricular function. Successful radiofrequency ablation of the arrhythmia resulted from application of energy to a single site at the posterior margin of the fossa ovalis in the right atrium. The investigators further noted that at times this arrhythmia exhibited minimal isoelectric baseline between the P waves, and thus may have more closely resembled atrial fibrillation. Recordings suggestive of atrial fibrillation in the context of MAT have been reported (1,5,6,15) and were seen in one of our patients (Fig. 4C). This raises the possibility that MAT may occasionally induce atrial fibrillation in a manner similar to the "focal" atrial fibrillation described in adults by Haïssaguerre et al (16).

MAT and respiratory infection. A number of patients with MAT have been diagnosed in the context of a respiratory infection, suggesting a possible link. The association between SVT and viral illness was first proposed several decades ago (17). However, little evidence is available to support a causal relationship. Terasaki et al. (18) described a variety of supraventricular and ventricular tachyarrhythmias in a murine coxsackie B3 myocarditis model.

Furthermore, Donnerstein et al. (15) described eight patients with respiratory syncytial virus (RSV) and atrial arrhythmias; four were diagnosed with automatic atrial arrhythmias versus chaotic atrial tachycardia. In the review by Wu et al. (9), 17 of 22 patients with MAT had respiratory illnesses. In our study, a significant number (29%) had intercurrent respiratory illness. Finally, the well-known association of MAT and chronic obstructive pulmonary

Table 1. Continued

Ventricular Rate (beats/min)	Echocardiographic Result	Current Health Status	Arrhythmia Duration (days)	Duration of Follow-up
253	NI function	Normal	148	5 yr 4 mo
208	SF 35%, EF 55%	Normal	291	19 mo
136	SF 31%, "dilated heart"	Normal	n/a	n/a
242	SF 24%	Normal	43	38 mo
125	Normal	Developmental delay, deaf	555	54 mo
200	Normal	Lost to follow-up	n/a	3 yr 9 mo
175	None	Normal	26	5 yr 4 mo
132	EF 57%	Normal	192	8 yr 6 mo
190	Normal ventricular function	Normal	360	10 yr 11 mo
146	None	PI, decreased RV fx, bifascicular block, deaf, pacemaker	112	10 yr 10 mo
111	None	Normal	102	21 mo
155	None	Asymptomatic, followed for HOCM	9	17 yr 9 mo
179	LVEDD 95 percentile, LVSD 95 percentile	Lost to follow-up	110	15 mo
210	Normal	Lost to follow-up	n/a	n/a
150	Normal	Normal	69	n/a
146	Dilated, poorly contractile LV	Normal	20	20 mo
230	HOCM, SF 51%, EF 88%	Asymptomatic, followed for HOCM	357	23 mo
	None	Normal	332	36 mo
170	None	Normal	8	24 mo
210	Normal LV and RV fx, PS with 45 mm gradient	Normal	n/a	2 mo
250	Normal LV fx, 3+ MR, dilated left atrium, 1+ TR	Deceased	244	n/a

disease in the adult patient suggests more than a coincidental relationship. Pulmonary disease may influence the electrophysiologic properties of the heart by such mechanisms as release of pulmonary inflammatory cytokines, the indirect effects of altered pulmonary mechanics and gas exchange, or direct myocardial inflammation, altering myocyte membrane properties and resulting in abnormal automaticity. As both our experience and the experience of others have shown, electrical cardioversion does not convert MAT to sinus rhythm, thus providing additional support for an automatic mechanism.

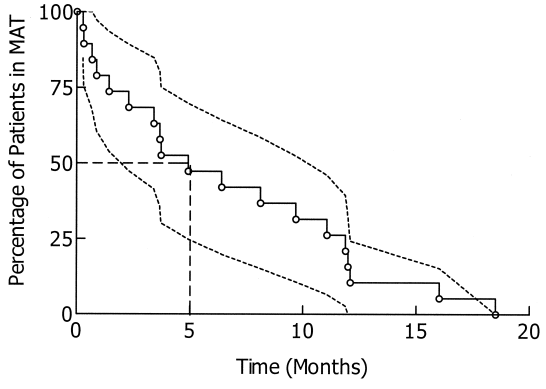

Figure 3. Kaplan-Meier analysis of duration of arrhythmia for the 19 patients in whom it was known. **Dotted lines** indicate the 95% confidence intervals. Fifty percent of patients reverted to sinus rhythm within five months. MAT = multifocal atrial tachycardia.

Incidence. The incidence of MAT in children is unknown, but it appears to be low. As suggested by our ten (48%) asymptomatic patients, there may be individuals who de-

Table 2. Attempted Treatment for MAT

Antiarrhythmic medication	15/21
Digoxin	14
Propranolol	4
Quinidine	3
Amiodarone	1
Esmolol	1
Lidocaine	1
Procainamide	1
Cardioversion	4/21
Synchronized direct current	3
Overdrive pacing	3

MAT = multifocal atrial tachycardia.

Table 3. Published Reports of MAT in children

Authors	Ref.	Year	n	Follow-up Interval	Comment
Farooki and Green	7	1977	2	3 yr	First report in children; fetal diagnoses
Bietzke	6	1979	1	14 mo	Atrial tachycardia, fibrillation, flutter and MAT in one infant; CHF at presentation
Bisset et al.	5	1981	10	1–36 mo	1 death
Southall et al.	19	1981	2	3 yr	Survey of rhythm in >3,000 newborns
Liberthson and Colan	1	1982	9	3–38 mo	Rx predominantly with digoxin, propranolol, quinidine
Wu et al.	9	1984	22	3.5 yr (avg)	CHD diagnoses by clinical examination; 3 deaths, 2 with SHD
Yeager et al.	11	1984	4	3–26 mo	Report proposing risk with MAT for sudden death; 3 deaths, 2 with SHD
Zeevi et al.	24	1985	1	6 mo	Patient with L-TGA. Control achieved after 16 hours of treatment with amiodarone
Chantepie et al.	25	1986	1	1 yr	WPW and MAT, Rx digoxin, disopyramide, and amiodarone
Esterl and Rösel	28	1987	1	8 yr	MAT attributed to intrapartum oxytocin; treated with digoxin
Strasburger et al.	22	1988	5	n/a	Report of encainide in various childhood SVTs; 1 sudden death attributed to treatment
Houyel et al.	23	1990	2	6–17 mo	Report of flecainide in MAT
Tipple and Sandor	21	1991	2	1–15 mo	Report of sotalol in childhood SVTs; 1 MAT case refractory to therapy
Maragnès et al.	20	1992	1	n/a	Report of sotalol in childhood SVTs
Donnerstein et al.*	15	1994	4	1–5 yr	Associated various SVT mechanisms with RSV infection
Dodo et al.	8	1995	9	25 mo (avg)	Rx procainamide, propafenone and Amio; 2 deaths, both SHD, attributed to sepsis and OHS
Fish et al.	12	1996	7	1–14 mo	Three patients with RSV; propafenone used in 6 of 7 patients
Salim et al.	10	1997	5	1–59 mo	One death, attributed to digoxin, encainide, atenolol treatment
Cetta et al.	29	1997	1	None	Treatment with digoxin/propranolol
Bevilacqua et al.	14	2000	1	3 mo	Successful radiofrequency ablation
Bradley et al.		2001	21	1–18 yr	Current study

*Four cases reported as "AAT vs. CAT."

AAT = automatic atrial tachycardia; avg = average; CAT = chaotic atrial tachycardia; CHD = congenital heart disease; CHF = congestive heart failure; L-TGA = levo-transposition of the great arteries; MAT = multifocal atrial tachycardia; n/a = not available; OHS = open heart surgery; RSV = respiratory syncytial virus; SHD = structural heart disease; SVT = supraventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

velop this arrhythmia and revert to sinus rhythm before it is detected. Southall et al. (19) prospectively evaluated ECGs of 3,383 normal newborns. Those with abnormalities underwent repeated 24-h Holter testing over their first three years of life. Two of the 3,383 newborns had MAT, both asymptomatic at three years of age. These data would suggest an incidence in neonates of 0.02%.

Therapy. There is no standard, proven therapeutic approach to patients with MAT. Wu et al. (9) saw little benefit to a variety of antiarrhythmic agents early in their experience, and they refrained from treating their later patients. The mechanism of the arrhythmia was initially believed to be re-entrant in four of our patients, and cardioversion was attempted (Table 2). It was uniformly unsuccessful, a hallmark of this arrhythmia. Digoxin, the most commonly used medication in our and other series, was given to slow the ventricular response.

The use of several newer antiarrhythmic agents has been described for children with MAT, with mixed results. In two series describing the use of sotalol for various pediatric SVTs, two out of three patients with MAT did not respond (20,21). Although encainide is no longer available, response to encainide in five children was either "partial(ly) effective" or "ineffective" (22). A report of two patients suggests that flecainide may be effective in restoring sinus rhythm (23). Fish et al. (12) described a response to propafenone within 48 h in five of six patients in whom it was used. Finally, amiodarone, used in one of our patients, has also been reported to be effective in MAT, and it is frequently credited with superior control of the arrhythmia compared to other agents (8,10,24,25). It should be noted that diverse effects of antiarrhythmic agents were thought to account for the poor outcomes of three patients in published series (Table 3) (8,10,22). Although it remains possible that diminished left ventricular function seen in some patients was due to tachycardia-induced cardiomyopathy (26,27), ventricular rate was not closely linked to functional compromise in our patients, and thus does not alone dictate a need for treatment. Our current practice is to treat underlying illness, and, when present, treat echocardiographically documented ventricular dysfunction with digoxin. Asymptomatic patients are followed without pharmacologic treatment, and medications such as amiodarone are reserved for symptomatic patients with persistent MAT.

Prognosis. The extended follow-up of this study confirms prior reported observations of patients with MAT, supporting the low mortality and likely good outcome. Long-term health appears related primarily to underlying conditions. Patients with normal cardiac anatomy and mild respiratory illness or no symptoms had spontaneous resolution with completely normal health status at follow-up. Those with congenital anomalies or clinical syndromes demonstrated chronic problems consistent with these diagnoses. Recurrence of MAT or other arrhythmias was not observed in any patient.

Figure 4. Selected electrocardiograms. **(A,B)** Typical findings of multifocal atrial tachycardia, demonstrating rapid atrial rate with multiple P-wave morphologies, and irregular, rapid ventricular response (patients 6 and 21). **(C)** Atrial fibrillation-like tracing (patient 5; see text).

Study limitations. The limitations of this study of MAT are several. The patient sample is retrospective and, therefore, biased toward children who had regular contact with the health care system. For example, monitoring for separate medical issues or at health maintenance visits identified three patients. Similarly, the closer monitoring inherent in the care of patients with heart disease may lead to preferential recognition of MAT in this group, and thus a disproportionate representation of them in our and other reports. Finally, estimates of onset, duration, and termination of arrhythmia are confounded by variable and infrequent sampling (i.e., ECGs obtained at follow-up intervals).

Conclusions. Most children with MAT are infants and are neither very ill at the onset of the arrhythmia nor adversely affected by it over the several months it typically persists. The condition may accompany mild respiratory illnesses or, less commonly, critical cardiac and pulmonary disease, but MAT is not a strong indicator of underlying heart disease. Even in patients without symptoms, mild ventricular dysfunction may be seen echocardiographically, but resolution is generally complete, without relapse. Response to antiarrhythmic agents is limited, although amiodarone shows promise when control of the rhythm is necessary. Direct current cardioversion is ineffective and should be avoided. Management should consist of periodic monitoring and, when necessary, care directed to underlying comorbid conditions. Finally, long-term cardiovascular and developmental outcome depends principally on the overall clinical state; for otherwise healthy children, it is excellent.

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